DIAGNOSTIC DILEMMA
Aimee K. Zaas, MD, Section Editor

THE AMERICAN JOURNAL of MEDICINE®

Pain at the Game: Spontaneous Coronary Artery Dissection

Gagan D. Singh, MD, Marin Nishimura, BS, Jason H. Rogers, MD, Ezra A. Amsterdam, MD
Division of Cardiovascular Medicine, Department of Internal Medicine, University of California Davis Health System, Sacramento, CA.

PRESENTATION

Some parents become rather impassioned at their children's sporting events, but for one patient, game day was extraordinarily traumatic. Shortly after arguing with another parent at her daughter's soccer match, a 34-year-old Caucasian woman began experiencing crushing and unrelenting substernal chest pressure. Two hours after the discomfort began, she sought medical attention at the emergency department. On presentation, she was dizzy and nauseous but denied diaphoresis, dyspnea, positional aggravation or alleviation of her pain, orthopnea, paroxysmal nocturnal dyspnea, or edema.

The patient had no history of smoking, heavy alcohol consumption, or illicit drug use. Her family medical history was notable for hypertension and cerebrovascular disease. A review of systems was pertinent for active menstruation at the time of presentation.

ASSESSMENT

On examination, the patient was anxious but in no acute distress. She was afebrile, her heart rate was 85 beats per minute, her blood pressure was 135/58 mmHg, her respiratory rate was 21 breaths per minute, and her oxygen saturation was 98% on room air. She had no jugular venous distension, and her lungs were clear to auscultation bilaterally. Her cardiac rhythm was regular without murmurs, clicks, or gallops, and the point of maximal impulse was nondisplaced. She had no lower extremity edema.

An electrocardiogram (ECG) demonstrated normal sinus rhythm, a normal QRS axis, and anterior upsloping ST segment elevations (**Figure 1A**), which resolved with sublingual nitroglycerin (**Figure 1B**). The patient had not had a prior ECG. A chest film showed a normal cardiac silhouette with clear lung fields and no other acute pathologic process. Laboratory data were notable for the following: white blood cell count, 11.5 x 10^3 cells/uL; hemoglobin, 10.4 g/dL; glucose, 134 mg/dL; troponin I, 0.13 ng/mL (reference range, 0-0.04 ng/mL); and D-dimer, 152 ng/mL (reference range, 0-230 ng/mL).

After the first evaluation, it was believed that the patient's symptoms were unlikely to have been caused by coronary plaque disruption, given her sex and age. On reevaluation 3 hours after presentation, the patient continued to complain of nausea but was free of chest pain. However, her troponin I value had risen to 50 ng/mL, and a repeat ECG revealed deep T-wave inversions in the anterolateral leads (**Figure 1C**). A cardiology consultation was obtained.

Based on the patient's history, presentation, ECG changes, and the trajectory of cardiac biomarkers, she underwent invasive coronary angiography. Her coronary angiogram showed no significant epicardial atherosclerotic coronary artery disease, congenital coronary anomalies, or any evidence of vasospasm. However, the distal left anterior descending artery was notable for an abrupt decrement in luminal diameter and a ribbon-like appearance suggestive of coronary artery dissection (**Figure 2**).

DIAGNOSIS

Some 0.2%-1.1% of patients undergoing angiography for acute coronary syndrome are found to have spontaneous coronary artery dissection.[1] An estimated 800 new cases of spontaneous coronary artery dissection occur in the United States each year.[1]

This atraumatic noniatrogenic separation of coronary artery layers by hemorrhage leads to formation of an intramural hematoma.[1] The first of 2 proposed mechanisms involves initiation of dissection and hemorrhage by an intimal

Funding: None.
Conflict of Interest: None.
Authorship: All authors had access to the data and a role in writing the manuscript.

Requests for reprints should be addressed to Ezra A. Amsterdam, MD, 4860 Y Street, Suite 2820, Sacramento, CA, 95817.
E-mail address: eaamsterdam@ucdavis.edu

http://dx.doi.org/10.1016/j.amjmed.2014.08.002

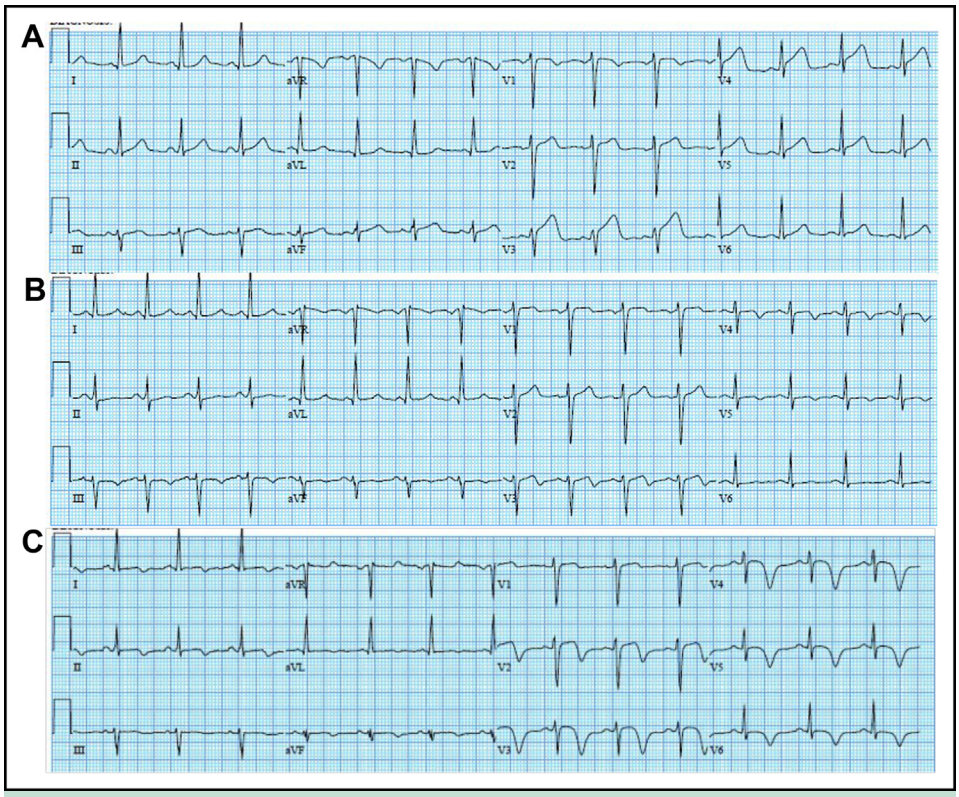

Figure 1 (**A**) an electrocardiogram (ECG) was obtained on presentation during active chest pain. (**B**) Another was ordered after administration of sublingual nitroglycerin, while the patient was free of discomfort. (**C**) Three hours after presentation, a third ECG was performed for reevaluation. The patient had no chest pain at this point.

tear and creation of a false lumen; much like classic aortic dissection. The second is rupture of the vasa vasorum, leading to intramural hemorrhage and medial dissection without intimal tearing.[1] Subsequent formation of an intramural hematoma could compress the true lumen, causing downstream myocardial ischemia and/or infarction.

Clinical presentation depends on the extent and severity of the dissection and the coronary artery involved. Presenting symptoms range from chest pain alone to frank acute coronary syndrome, ventricular fibrillation, and/or sudden death.[1,2] Multiple series of patients with spontaneous coronary artery dissection illustrate its strong predilection for otherwise healthy young women.[3-5] Of those who suffer spontaneous coronary artery dissection, 70-82% are women, and the mean age of all patients, male and female, is 40-43 years.[3,4] Among women younger than 50 years who present with acute coronary syndrome, the prevalence of spontaneous coronary artery dissection is 9%.[1]

Connective tissue disorders, coronary artery spasm, strenuous exercise, emotional stress, peripartum/postpartum status, use of oral contraceptive pills, and menstruation have all been associated with spontaneous coronary artery dissection.[1,6] Recently, fibromuscular dysplasia has also been identified as an independent predictor, implying that underlying systemic vascular abnormalities predispose coronary arteries to dissection, especially in conjunction with precipitating factors such as emotional stress.[6]

Our patient's young age, noncontributory past medical history, and the temporal relationship of chest pain to emotional distress may suggest an initial noncoronary chest pain syndrome. However, dynamic ECG changes and positive troponin levels in such patients should prompt clinicians to investigate nonatherosclerotic causes of coronary ischemia, such as coronary dissection, embolism, or vasospasm. Coronary angiography confirmed that our patient had spontaneous coronary artery dissection of the left anterior descending artery. In many other situations, spontaneous coronary artery dissection is poorly characterized by angiography, as the narrowing caused by the intramural hematoma can be misinterpreted as atherosclerotic disease.[7] However, intracoronary imaging, such as intravascular ultrasound and optical coherence tomography, are powerful tools in making a definitive diagnosis (**Figure 3**).[7]

MANAGEMENT

The optimal treatment strategy for spontaneous coronary artery dissection is not well defined and hinges on the clinical presentation, extent of dissection, and amount of